American Journal of Alzheimer's Disease & Other Dementias®
2015, Vol. 30(2) 173-177

DOI: 10.1177/1533317514542642
aja.sagepub.com
SAGE

Clinical Characterization of Mild Cognitive Impairment as a Prodrome to Dementia With Lewy Bodies

Christine M. Belden, PsyD[1], Vickram Kahlon[1], Michael Malek-Ahmadi, MSPH[1], Andrew Tsai[1], and Marwan N. Sabbagh, MD[1]

Abstract

Limited information regarding the specificity of mild cognitive impairment (MCI) as it relates to dementia with Lewy bodies (DLB) exists. Here, we summarize the clinical phenotype of MCI in clinically suspect DLB. Ten patients with a primary diagnosis of MCI and secondary diagnoses of DLB were identified. Patients underwent clinical neurological and neuropsychological evaluation that included application of McKeith criteria. We found parkinsonism and gait abnormality in 9 of the 10 patients; fluctuations in 8 of the 10; and hallucinations and dream enactment behavior in 5 of the 10. Of the 10 cases, 4 were classified as nonamnestic MCI and 6 were amnestic MCI. Of the 10 cases, 9 displayed executive and/or visuospatial dysfunction. Of the 10 cases, 6 have progressed to DLB. Progression of MCI to DLB is not dependent on memory impairment. The presence of core clinical features—parkinsonism and cognitive fluctuations—and predominant executive and visuospatial dysfunction $\pm$ memory impairment is suggestive of a prodromal DLB presentation.

Keywords

mild cognitive impairment, dementia with Lewy bodies, unique clinical features, neuropsychological–cognitive profiles

Introduction

Mild cognitive impairment (MCI) is considered a transitional state between cognitive changes in normal aging and early stage dementia.[1] Since publication of the original Petersen criteria, MCI has come to be recognized as extending beyond memory impairment and has been refined to include amnestic and nonamnestic types.[2] Previous studies[3-7] suggest that amnestic MCI is most likely to progress to Alzheimer's dementia (AD), whereas nonamnestic MCI is more heterogeneous in terms of rate of progression and progression end points. Nonamnestic MCI may progress to vascular dementia, frontotemporal dementia, dementia with Lewy bodies (DLB), and others.[3-7]

Currently, limited information is available regarding the specificity of MCI in relation to DLB. Dementia with Lewy bodies is characterized by a progressive dementia with predominant deficits on measures of attention, executive functions, and visuospatial abilities; memory impairment may not occur in the earlier stages.[8-12] Core features include fluctuations in cognition with pronounced variation in attention/alertness, recurrent, well-formed visual hallucinations, and spontaneous parkinsonism.[13] Suggestive features include rapid eye movement (REM) sleep behavior disorder (RBD)[14] and severe neuroleptic sensitivity.[15] Additional neuropsychiatric features supportive of DLB include hallucinations in other modalities, delusions, and misidentification syndromes (Capgras syndrome, phantom border, and reduplication of person or place[16,17]).

Previous research has suggested that individuals with MCI associated with specific clinical features are at increased risk of progression to DLB. Ferman et al[18] found that in longitudinally followed patients with MCI, those with nonamnestic MCI were 10 times more likely to develop clinically probable DLB; conversely, those with amnestic MCI were 10 times more likely to develop clinically probable AD. Of those with clinically probable DLB, 88% presented initially with attention and/or visuospatial impairment; 24.5% of this group had concomitant memory impairment. Further, these patients were more likely to have RBD, greater daytime sleepiness, and greater likelihood of fluctuations in the MCI state. Molano et al[19] found autopsy-confirmed DLB in all cases with RBD

[1] The Cleo Roberts Center for Clinical Research, Banner Sun Health Research Institute, Sun City, AZ, USA

Corresponding Author:
Marwan N. Sabbagh, MD, The Cleo Roberts Center for Clinical Research, Banner Sun Health Research Institute, 10515 West Santa Fe Drive, Sun City, AZ 85351, USA.
Email: marwan.sabbagh@bannerhealth.com

Table 1. Demographic Data of Individual Cases.

	Age	Gender	Education	Disease Duration
Case 1	65	M	17	5
Case 2	71	F	12	2
Case 3	75	M	13	1
Case 4	73	M	20	6
Case 5	78	M	16	11
Case 6	64	M	14	4
Case 7	69	M	19	2.5
Case 8	70	M	16	1.5
Case 9	84	M	16	2
Case 10	74	M	16	3

Abbreviations: F, female; M, male.

and MCI (any subtype). Attention, executive functioning, and visuospatial functions were most commonly affected. Jicha et al[20] found that neuropathologically confirmed cases with MCI-DLB and MCI-AD differed clinically in the expression of Parkinsonism, provoked hallucinations or delirium, or the presence of any of the noncognitive symptoms of DLB. Further, letter fluency and narrative recall performance were significantly different between the groups.[21-23] As such, the purpose of this study is to summarize the clinical phenotype of MCI in the setting of clinically suspect DLB.

Methods

Patients

Patients were selected via query of the Banner Sun Health Research Institute memory disorder clinic patient database. Patients identified were diagnosed, using *International Classification of Diseases, Ninth Revision* codes, as 331.83 (MCI) primary and 331.82 (DLB) secondary (indicating suspected etiology underlying MCI). Information pertaining to each case was gathered via retrospective chart review as approved by the Banner institutional review board. A total of 10 patients (9 males and 1 female) were identified and ranged in age from 64 to 84 with a mean of 72.30 $\pm$ 5.96 years. Patients ranged in years of education from 12 to 20 years with a mean of 15.90 $\pm$ 2.47 years. Patients ranged in duration of disease from 1 to 11 years with a mean of 3.80 $\pm$ 2.98 years. Patient demographic data can be found in Table 1.

Clinical Assessment

Specific clinical features examined in this study include fluctuations, hallucinations, dream enactment behavior (RBD), gait abnormalities, parkinsonism, and impaired cognitive domains. A neurologist (MS) specializing in dementia and cognitive disorders evaluated each patient through comprehensive neurological examination. Clinical diagnosis of MCI subtype was based on criteria set forth by Petersen et al.[2,3] Clinical diagnosis of suspect DLB was based on criteria set forth by McKeith et al.[13] Patients and/or their family were questioned regarding presence of clinical features (fluctuations, hallucinations, dream enactment behavior, and parkinsonism), and presence of parkinsonism and/or gait abnormalities were noted if present. All patients underwent comprehensive neuropsychological evaluation that included measures of verbal recall, visual recall, language, visuospatial skills, executive functions, psychomotor processing speed, and attention/concentration. Disease progression was tracked to determine which individuals went on to meet criteria for clinically suspect DLB.

Neuropsychological Assessment

Seven patients underwent outpatient neuropsychological evaluation within our Memory Disorders Clinic. In all, 2 were evaluated by outside neuropsychologists, and 1 was evaluated as part of a Brain and Whole Body Donation project within the Banner Sun Health Research Institute. All neuropsychological evaluations were reviewed by 1 neuropsychologist (CMB) specializing in dementia and cognitive disorders to confirm domains of impairment and cognitive diagnosis.

Tests administered included assessment of global intelligence (Wechsler Adult Intelligence Scale—Third Edition [WAIS-III],[24] Wechsler Abbreviated Scale of Intelligence [WASI],[25] and Wechsler Adult Intelligence Scale—Fourth Edition [WAIS-IV][26]), memory (Logical Memory Subtest of the Wechsler Memory Scale Revised[27] Logical Memory and Visual Reproductions subtests of the Wechsler Memory Scale—Third Edition,[28] Brief Visual Memory Test Revised,[29] Rey-Osterreith Complex Figure Recall,[30,31] Rey Auditory Verbal Learning Test,[32] California Verbal Learning Test—Second Edition,[33] and immediate and delayed recall tasks from Repeatable Battery for the Assessment of Neuropsychological Status [RBANS][34]), language functioning (Boston Naming Test—Second Edition,[35] Controlled Oral Word Association Test,[36] category/semantic fluency [animals],[37] and language tasks from RBANS[34]), visuospatial/perceptual functioning (Rey-Osterreith Complex Figure,[30,31] Judgment of Line Orientation,[38] RBANS copy and judgment of line orientation tasks[34] Block Design from WAIS-III,[24] WAIS-IV,[26] and/or WASI,[25] and Visual Puzzles from WAIS-IV[26]), attention (Trail Making Test A,[39] Digit Span from WMS-R,[27] WAIS-III/IV,[24,26] or RBANS,[34] Digit Symbol subtest from Wechsler Adult Intelligence Test-Revised [WAIS-R][39] or WAIS-IV[26]), and executive functioning (Trail Making Test B,[40] Stroop Color/Word,[41] and Wisconsin Card Sorting Test[42]). Additional measures include the Mini-Mental State Examination,[43] Clock Drawing Test,[44] and Blessed Orientation-Concentration-Memory Test.[45,46]

Results

Prospectively applying McKeith criteria[13] identified 10 patients with MCI clinically suspected to be a prodrome of DLB. The case series is comprised of 9 males and 1 female. Average age is 72.30 $\pm$ 5.96 years with an average education level of 15.90 $\pm$ 2.47 years. Average disease duration was 3.80 $\pm$ 2.98 years. The presence of RBD, fluctuations,

Table 2. Clinical Characteristics of Individual Cases.

	RBD	Parkinsonism	Fluctuations	Hallucinations	Progression to Clinically Suspect DLB	Gait Abnormality
Case 1	Absent	Present	Present	Absent	DLB	Present
Case 2	Absent	Present	Present	Absent	DLB	Present
Case 3	Absent	Present	Present	Present	DLB	Absent
Case 4	Present	Present	Absent	Absent	DLB	Present
Case 5	Present	Present	Present	Present	No	Present
Case 6	Present	Present	Present	Present	DLB	Present
Case 7	Absent	Present	Present	Absent	No	Present
Case 8	Present	Present	Present	Absent	DLB	Present
Case 9	Absent	Present	Present	Present	No	Present
Case 10	Present	Absent	Absent	Present	No	Present

Abbreviations: DLB, dementia with Lewy Bodies; RBD, rapid eye movement sleep behavior disorder.

Table 3. Neuropsychological Assessment of Individual Cases.

	Neuropsychological Profile
Case 1	Nonamnestic, single-domain MCI (executive)
Case 2	Nonamnestic, multiple-domain MCI (visuospatial, executive)
Case 3	Amnestic, multiple-domain MCI (visuospatial, attention, executive)
Case 4	Nonamnestic, single-domain MCI (visuospatial)
Case 5	Amnestic, multiple-domain MCI (executive, visuospatial)
Case 6	Amnestic, multiple-domain MCI, (visuospatial, executive)
Case 7	Amnestic, multiple-domain MCI, (visuospatial, executive)
Case 8	Nonamnestic, multiple-domain MCI (visuospatial, executive)
Case 9	Amnestic, multiple-domain MCI (visuospatial, executive)
Case 10	Amnestic, multiple-domain MCI (visuospatial, executive)

Abbreviation: MCI, mild cognitive impairment.

hallucinations, parkinsonism, gait abnormality, MCI subtype, and domains of impairment are reported in Tables 2 and 3. Parkinsonism and gait abnormality were the most prevalent clinical features as both manifested in 9 of the 10 cases. Fluctuations were found in 8 of the 10 cases, and hallucinations and dream enactment behavior were noted in 5 of the 10 cases.

Neuropsychological evaluation classified 4 of the 10 cases as nonamnestic MCI and 6 of the 10 as amnestic MCI. Of the 4 nonamnestic cases, 2 were single domain (1 case = executive only and 1 case = visuospatial only) and 2 were multidomain (executive and visuospatial). All 6 amnestic cases were multidomain. Five cases had executive and visuospatial dysfunction in addition to memory impairment. The sixth case had executive, visuospatial, and attention impairment. In total, executive and visuospatial impairment was noted in 9 of the 10 cases. In all, 6 cases had progressed to full-onset DLB at the time of this report; prodromally, 2 of these cases were classified as nonamnestic MCI (1 executive and 1 visuospatial), 2 had nonamnestic multidomain MCI (visuospatial and executive), and 2 had amnestic multidomain MCI (visuospatial and executive).

Discussion

In this study, we find that parkinsonism and cognitive fluctuations were the most common core DLB features to appear in the MCI state. Furthermore, we find that the cognitive profile of MCI in the context of DLB is characterized primarily by executive and visuospatial dysfunction with variability in memory.

RBD and hallucinations, although frequent, are less ubiquitous than parkinsonism and fluctuations in our cases series. Molano et al[19] found fluctuations and parkinsonism in 6 of the 8 and 8 of the 8 cases, respectively; fluctuations were present in 8 of the 10 of our cases, and parkinsonism was present in 9 of the 10 cases. Hallucinations and dream enactment behavior were present in 5 of the 10 of our cases as compared to Molano et al[19] (6 of the 8 cases). In their retrospective analysis, Jicha et al[20] found hallucinations in 4 of the 9, fluctuations in 3 of the 9, and parkinsonism in 5 of the 9 cases with MCI-DLB, but in none of their cases with MCI-AD. In their longitudinal analysis, Ferman et al[18] found greater likelihood of RBD, greater overall daytime sleepiness, and greater likelihood of fluctuations in their MCI-DLB sample.

The cognitive profile of MCI in the setting of AD is characterized primarily by an impairment of episodic memory.[1-3,14] Dysfunction in other domains, that is, language, executive, attention, and visuospatial, may also be present in word-finding difficulties, spatial cognition deficits, and impaired problem solving and judgment.[11,23,47] In contrast, the cognitive profile of DLB is characterized primarily by dysfunction of attention, executive, and visuospatial skills.[9-12]

Consistent with previous research,[9-12] neuropsychological assessment in our case series revealed predominance of visuospatial and executive dysfunction. Of the 10 cases, 9 had executive dysfunction and 9 of the 10 cases had visuospatial dysfunction; 8 of the 10 cases had both executive and visuospatial dysfunctions. Both amnestic and nonamnestic subtypes progressed to clinically suspect DLB. The majority of our cases have memory impairment (6 of 10). Of the 6 cases that progressed to full-onset, clinically suspect DLB, 4 were nonamnestic cases in the prodromal phase. The findings of this case series are similar to those of Molano et al,[19] who concluded that both amnestic and nonamnestic MCI subtypes progress to DLB and that impairments in executive functions and visuospatial skills were most common. Jicha et al[20] also determined that early memory loss may lack specificity for differentiating early AD from early DLB. In contrast, Ferman

et al[18] found that nonamnestic MCI patients were 10 times more likely to develop clinically probable DLB as compared to AD. However, 88% of those who progressed to clinically probable DLB initially presented with attention and/or visuospatial impairment and 24.5% of that group had concomitant memory difficulty. Our results lend further support to the presence of a prodromal MCI stage of DLB marked by specific clinical and neurocognitive features. Further, MCI-DLB had a high rate of progression to full–onset, clinically suspect DLB.

The strengths of this study include the comprehensive neuropsychological evaluation by which MCI subtype was determined. Although cognitive evaluation conducted by multiple individuals is a weakness, a single neuropsychologist reviewed each evaluation to confirm MCI diagnosis and domains of impairment thus improving diagnostic reliability. Lack of pathological confirmation of DLB is a weakness of this study. However, a single neurologist (MS) specializing in cognitive disorders examined each patient and prospectively applied established DLB diagnostic criteria[13] in his clinical examination. Although formal measures of core, noncognitive criteria were not utilized, a single expert clinician (MS) examined each patient and recorded presence or absence of symptoms for each patient based upon information provided by the patient and family members, as well as clinical observations. Because diagnoses and observations were noted in real time prior to the initiation of this retrospective study, bias is effectively removed. Jicha et al[20] utilized a retrospective application in their study of neuropathologically confirmed DLB and found this approach to be feasible when adequate prospectively collected data exist. Additional weaknesses lie in the small sample size, lack of longitudinal data, and a relatively heterogeneous sample (predominantly Caucasian, male, and highly educated population).

Despite these limitations, this case series contributes to the current (albeit limited) research on MCI as a prodrome to DLB.[19,20] Our case series demonstrates that progression of MCI to DLB is not dependent on the absence of early memory impairment/amnesia. Rather, diagnosis of MCI as a prodrome to DLB can be made in the presence of core clinical features—namely parkinsonism and cognitive fluctuations. Additionally, cognitive impairment with predominant executive and visuospatial dysfunction, with or without the presence of memory impairment, is suggestive of a prodromal DLB presentation.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This research is supported by the Banner Sun Health Research Institute and the National Institute on Aging (P30 AG19610).

References

1. Petersen RC, Smith GE, Waring SC, Ivnik RJ, Tangalos EG, Kokmen E. Mild cognitive impairment: clinical characterization and outcome. *Arch Neurol*. 1999;56(3):303-308.
2. Petersen RC, Negash S. Mild cognitive impairment: an overview. *CNS Spectr*. 2008;13(1):45-53.
3. Petersen RC. Mild cognitive impairment as a diagnostic entity. *J Intern Med*. 2004;256(3):183-194.
4. Albert MS, DeKosky ST, Dickson D, et al. The diagnosis of mild cognitive impairment due to Alzheimer's disease: recommendations from the National Institute on Aging and the Alzheimer's Association workgroup. *Alzheimers Dement*. 2011;7(3):270-279.
5. Busse A, Hensel A, Guhne U, Angermeyer MC, Riedel Heller SG. Mild cognitive impairment: long-term course of four clinical subtypes. *Neurology*. 2006;67(12):2176-2185.
6. Fischer P, Jungwirth S, Zehetmayer S, et al. Conversion from subtypes of mild cognitive impairment to Alzheimer dementia. *Neurology*. 2007;68(4):288-291.
7. Lopez OL, Becker JT, Jagust WJ, et al. Neuropsychological characteristics of mild cognitive impairment subgroups. *J Neurol Neurosurg Psychiatry*. 2006;77(2):159-165.
8. Tiraboschi P, Salmon DP, Hansen LA, et al. What best differentiates Lewy body from Alzheimer's disease in early-stage dementia? *Brain*. 2006;129(pt 3):729-735.
9. Troster AI. Neuropsychological characteristics of dementia with Lewy bodies and Parkinson's disease with dementia: differentiation, early detection, and implications for 'mild cognitive impairment' and biomarkers. *Neuropsychol Rev*. 2008;18(1):103-119.
10. Connor DJ, Salmon DP, Sandy TJ, Galasko D, Hansen LA, Thal LJ. Cognitive profiles of autopsy-confirmed Lewy body variant vs pure Alzheimer disease. *Arch Neurol*. 1998;55(7):994-1000.
11. Ferman TJ, Smith GE, Boeve BF, et al. DLB fluctuations: specific features that reliably differentiate DLB from AD and normal aging. *Neurology*. 2004;62(2):181-187.
12. Oda H, Yamamoto Y, Maeda K. Neuropsychological profile of dementia with Lewy bodies. *Psychogeriatrics*. 2009;9(2):85-90.
13. McKeith IG, Dickson DW, Lowe J, et al. Diagnosis and management of dementia with Lewy bodies: third report of the DLB consortium. *Neurology*. 2005;65(12):1863-1872.
14. Murray ME, Ferman TJ, Boeve BF, et al. MRI and pathology of REM sleep behavior disorder in dementia with Lewy bodies. *Neurology*. 2013;81(19):1681-1689.
15. Ballard C, Aarsland D, Francis P, Corbett A. Neuropsychiatric symptoms in patients with dementias associated with cortical Lewy bodies: pathophysiology, clinical features, and pharmacological management. *Drugs Aging*. 2013;30(8):603-611.
16. Nagahama Y, Okina T, Suzuki N, Matsuda M, Fukao K, Murai T. Neural correlates of psychotic symptoms in dementia with Lewy bodies. *Brain*. 2010;133(pt 2):557-567.
17. Thaipisuttikul P, Lobach I, Zweig Y, Gurnani A, Galvin JE. Capgras syndrome in dementia with Lewy bodies. *Int Psychogeriatr*. 2013;25(5):843-849.
18. Ferman TJ, Smith GE, Kantarci K, et al. Nonamnestic mild cognitive impairment progresses to dementia with Lewy bodies. *Neurology*. 2013;81(23):2032-2038.

19. Molano J, Boeve B, Ferman T, et al. Mild cognitive impairment associated with limbic and neocortical Lewy body disease: a clinicopathological study. *Brain*. 2010;133(pt 2):540-556.
20. Jicha GA, Schmitt FA, Abner E, et al. Prodromal clinical manifestations of neuropathologically confirmed Lewy body disease. *Neurobiol Aging*. 2010;31(10):1805-1813.
21. Bradley BF. Dementia with Lewy bodies. *Dementia Continuum*. 2004;10(1):81-112.
22. Iachini T, Iavarone A, Senese VP, Ruotolo F, Ruggiero G. Visuospatial memory in healthy elderly, AD and MCI: a review. *Curr Aging Sci*. 2009;2(1):43-59.
23. Weintraub S, Wicklund AH, Salmon DP.The neuropsychological profile of Alzheimer disease. *Cold Spring Harb Perspect Med*. 2012;2(4):a006171.
24. Wechsler D. *Wechsler Adult Intelligence Scale-III*. San Antonio, TX: The Psychological Corporation; 1997.
25. Wechsler D. *Wechsler Abbreviated Scale of Intelligence*. San Antonio, TX: The Psychological Corporation; 1999.
26. Wechsler D. *Wechsler Adult Scale of Intelligence—Fourth Edition*. San Antonio, TX: The Psychological Corporation; 2008.
27. Wechsler D. *Wechsler Memory Scale-Revised: Manual*. San Antonio, TX: The Psychological Corporation; 1987.
28. Wechsler D. *Wechsler Memory Scale—Third Edition*. San Antonio, TX: The Psychological Corporation; 1999.
29. Benedict RHB. *Brief Visuospatial Memory Test-Revised*. Odessa, FL: Psychological Assessment Resources, Inc.; 1997.
30. Rey A. L'examen psychologique dans les cas d'encephalopathie traumatique.(Les problems.). *Arch Psychol*. 1941;28: 215-285.
31. Osterrieth PA. Filetest de copie d'une figure complex: Contribution a l'etude de la perception et de la memoire [The test of copying a complex figure: a contribution to the study of perception and memory]. *Arch Psychol*. 1944;30:286-356.
32. Rey A. *L'examen clinique en psychologie*. Paris: Presses Universitaires de France; 1958.
33. Dean CD, Joel HK, Edith K, Beth AO. *California Verbal Learning Test—Second Edition*. San Antonio, TX: The Psychological Corporation; 2000.
34. Christopher R. *Repeatable Battery for the Assessment of Neuropsychological Status—Form A*. San Antonio, TX: The Psychological Corporation; 2012.
35. Kaplan E, Goodglass H, Weintraub S. *Boston Naming Test—Second Edition (BNT-2)*. Baltimore: Lippincott Williams & Wilkins; 2001.
36. Benton AL, Hamsher K. *Multilingual Aphasia Examination*. Iowa City, Iowa: University of Iowa; 1989.
37. Tombaugh TN, Kozak J, Rees L. Normative data stratified by age and education for two measures of verbal fluency: FAS and animal naming. *Arch Clin Neuropsychol*. 1999;14(2):167-177.
38. Benton AL, Hannay HJ, Varney NR. Visual perception of line direction in patients with unilateral brain disease. *Neurology*. 1975;25(10):907-910.
39. Wechsler D. *Wechsler Adult Intelligence Scale-Revised*. San Antonio, TX: The Psychological Corporation; 1981.
40. Reitan RM, Wolfson D. *The Halstead-Reitan Neuropsychological Test Battery: Theory and Clinical Interpretation*. 2nd ed. South Tucson, AZ: Neuropsychology; 1993.
41. Golden CJ. *Stroop Color and Word Test*. Chicago IL: Stoelting; 1978.
42. Psychological Assessment Resources. *Computerised Wisconsin Card Sort Task Version 4 (WCST)*. Odessa, FL: Psychological Assessment Resources; 2003.
43. Folstein MF, Folstein SE, McHugh PR. Mini-mental state. *J Psychiatric Res*. 1975;12(3):189-198.
44. Borod JC, Goodglass H, Kaplan E.Normative data on the Boston diagnostic aphasia examination, parietal lobe battery and the Boston naming test. *J Clin Neuropsychol*. 1980;2:209-216.
45. Katzman R, Brown T, Fuld P, Peck A, Schechter R, Schimmel H. Validation of a short orientation-memory-concentration test of cognitive impairment. *Am J Psychiatry*. 1983;140(6):734-739.
46. Thomas H. *Six-Item Cognitive Impairment Test—Version 7*. Swindon, UK: Kingshill Research Centre; 2010.
47. McKhann G, Drachman D, Folstein M, et al. Clinical diagnosis of Alzheimer's disease: report of the NINCDS-ADRDA work group under the auspices of department of health and human services task force on Alzheimer's disease. *Neurology*. 1984;34(7):939-944.